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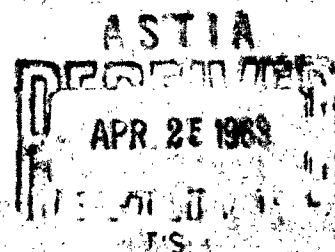# STUDIES IN DECOMPRESSION SICKNESS
Circulatory and Respiratory Changes Associated with Decompression Sickness in Anesthetized Dogs

FOREWORD

This report was prepared by the following personnel in the Physiology Branch:

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ABSTRACT

Twenty-four dogs were utilized in evaluating physiologic effects of intravascular bubbles. High and low atmospheric pressures were used, as well as direct introduction of air bubbles into the circulation. In all cases, the signs appearing as the result of intravascular bubbles were (1) marked tachypnea and (2) marked pulmonary arterial hypertension. Bubbles appeared first in the venous circulation, and were not seen in the arterial circulation until near collapse. Bubbles produced at altitude did not completely disappear on returning to ground level.

This technical documentary report has been reviewed and is approved.

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STUDIES IN DECOMPRESSION SICKNESS

Circulatory and Respiratory Changes Associated with Decompression Sickness in Anesthetized Dogs

1. INTRODUCTION

The etiology, symptoms, prognosis, and treatment of decompression sickness ("caisson disease") has been the subject of numerous research efforts since it was first described by Bert in 1878 (1). In attempting to compare the problem observed in altitude sickness (dysbarism, aero-embolism) with that of "caisson disease," it is obvious that a simple one-for-one relationship does not exist. The difficulty appears to lie in understanding the etiology of symptoms which occur when an organism is exposed to the same pressure gradient but in opposite directions. For example, permanent spinal cord lesions are very rarely seen in altitude sickness (2); whereas, they are quite common in decompression sickness (3).

Until recently, it was necessary to infer from the onset of tachypnea that bubbles might have accumulated and started to block the flow of blood through the lungs (7). This would tend to delay the spontaneous recovery from decompression sickness, since the lungs are the chief route for elimination of gases. Thus, if intravascular bubbles were to be formed more rapidly than they could be eliminated, and if they blocked the flow of lung blood, recovery would be impossible unless the bubbles were removed by contraction under higher atmospheric pressures or by redissolving in blood.

The purpose of this paper is to investigate some of the circulatory and respiratory adjustments associated with a change in ambient pressure, both hyperatmospheric and hypoatmospheric, on an experimental animal. The signs will be referred to hereafter as those resulting from decompression sickness as defined by Adler (4); therefore, decompression from high positive pressure and decompression to altitude will be treated as equal entities.[1]

2. METHODS

Compression-decompression studies

Sixteen mongrel dogs, weighing from 8.2 to 23.6 kg. individually, were anesthetized with pentobarbital sodium (Nembutal, 30 mg./kg., I.V.) with maintenance doses administered as required. Arterial and venous pressures were measured by Statham $P_{23}D_b$ strain gage transducers and recorded with a Sanborn 6-channel recorder (Model 150). A small hole drilled in the side of each transducer allowed for pressure equilibration of the diaphragm. Boiled, heparinized saline filled the transducers and catheters. Polyethylene catheters were threaded through the right femoral artery and vein for measurement of aortic and central venous pressures.

A double polyvinyl catheter similar to that described by Lategola (5) was used to monitor pulmonary artery pressure. The internal catheter, which was threaded through the pressure-sensing catheter and sealed against leakage, had a small balloon located at the tip. The double catheter was threaded through the right external jugular vein into the right ventricle. After entry into the right ventricle

[1] Decompression sickness is a syndrome, exclusive of hypoxia and airsickness, resulting from a reduction in barometric pressure.

was achieved, the balloon was inflated. When systolic contraction of the ventricle swept the catheter tip into the pulmonary artery, the balloon was deflated, leaving only the obstruction offered by the sensor tip in the pulmonary artery. A visual presentation of pressure changes was monitored on a Hewlett-Packard oscilloscope (Model 122AR) to assure placement of the catheter tip in the pulmonary artery. In four preparations, a No. 8 FR cardiac catheter was inserted through the left carotid artery into the left ventricle, and, subsequently, into the left atrium. Again, proper placement was achieved by monitoring pressure changes on the oscilloscope. Ventilation was accomplished by a tracheotomy in 13 animals and through an endotracheal tube in the remainder.

In 3 animals, two viewing cuvettes, similar to Armstrong's (6), were utilized in a left femoral vein-to-vein and left femoral artery-to-artery shunt. The cuvettes consisted of two Plexiglas blocks, 76 mm. by 19 mm., separated by a 1-mm.-thick polyethylene sheet. A section measuring 56 by 16 mm., tapered at both ends, was cut out of the polyethylene sheet, thus creating a space between the two blocks through which blood could flow. Openings were cut at each end of the block and appropriate connectors were attached. Twenty machine screws sealed the two blocks together. Polyethylene catheters then completed the arrangement so that blood could flow from a peripheral venous vascular bed through one cuvette in a thin film 1 mm. thick, and thence into the central circulation. In a similar manner, systemic arterial blood flowed through another cuvette and thence into a peripheral bed. The entire system was filled with boiled, heparinized normal saline before the connecting catheters were inserted. All animals were heparinized (5 mg./kg.) to preclude clotting in the viewing cuvettes or in the catheters. By using this method, the formation, location, and progress of bubbles could be seen on both the venous and arterial sides.

The chamber utilized in this study (fig. 1) consisted of a cylindric steel structure divided into a main compartment and a lock, each approximately 800 cu. ft. in volume with a 6.5-ft. round door as its single entry. Both sections, jointly or independently, had a pressure-range capability extending between 6 and 0.01 atmospheres absolute. Six observation windows were located at various points on the chamber.

The animal, with catheters in place, was moved into the main compartment of the chamber assembly, and the catheters were attached to appropriate transducers. Lead II ECG connections and a pneumograph were also attached. All connections led to an electrical panel box located outside the chamber, and the two were interconnected by means of sealed junctions. Thus, the desired physiologic parameters could be continuously monitored from the outside.

The compression-decompression schedule consisted of a 60-minute exposure to 6 atmospheres absolute pressure (75 p.s.i. gage) and decompression to ground level at the approximate rate of 0.5 atmosphere per minute. The animal was maintained at 1 atmosphere absolute pressure following decompression for a period varying between 30 to 45 minutes (hereafter referred to as ground +) and was then exposed to subatmospheric pressures. The ambient pressure of these subatmospheric exposures varied between 0.69 and 0.50 atmospheres (10,000 to 18,000 ft. equivalent) and was achieved at an ascent rate of 5,000 ft./minute. In all cases of altitude exposure, the animals were maintained on 100% O_2 to prevent hypoxia from interfering with signs of decompression sickness.

Decompression studies

Five mongrel dogs, weighing from 18.6 to 31.4 kg., were utilized in this series. Pulmonary artery pressure, systemic artery pressure, central venous pressure, ECG, and respiratory rate were recorded as described earlier. A plastic viewing cuvette was utilized in a left femoral vein-to-vein shunt in all animals and in a left femoral artery-to-artery shunt in 3 of the 5 animals. All were exposed
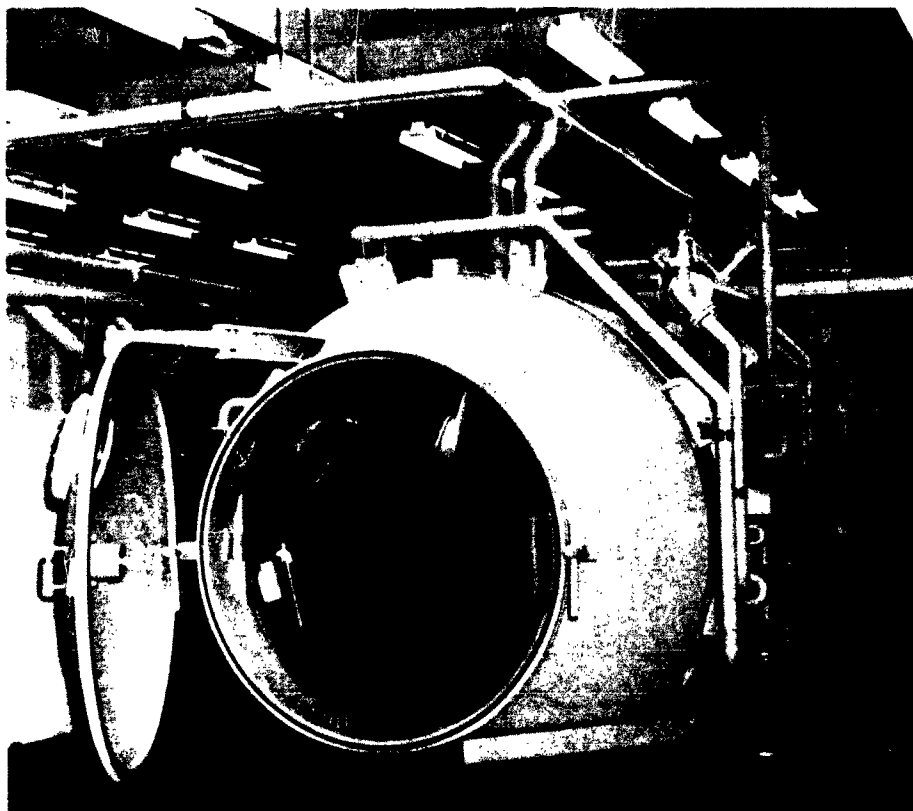
FIGURE 1

Combination high- and low-pressure chamber showing construction of reversible doors to lock and main compartments which withstand as much as 6 atmospheres and as little as 0.01 atmosphere absolute pressure.

to 0.18 atmosphere pressure (40,000 ft. equivalent), ascending at the rate of 5,000 ft./minute. The animals breathed 100% O_2 just prior to ascent and were maintained in this manner throughout the entire altitude procedure by use of a standard A-14 pressure-demand regulator.

Direct infusion

Three additional dogs had small bubbles infused at the rate of 5 cc./minute through a glass cannula inserted in the left femoral vein. A glass virus filter at the tip of the cannula regulated the bubble size to a diameter of 0.5 mm. Pulmonary artery pressure was obtained in only 1 animal. In the other 2, right

intraventricular pressure was recorded which gives evidence of changes in pulmonary arterial systolic pressure. Twenty-one, 30, and 47 cc. of air were bubbled into the bloodstreams of the respective dogs at ground level. The animal in which 47 cc. of air was bubbled was then taken from ground level to 0.69 atmosphere (10,000 ft.) in the low-pressure chamber.

3. RESULTS

Compression-decompression series

During compression and for a short period following decompression, there were no demonstrable abnormalities in the respiratory or in the cardiovascular systems as shown by that portion of the data in table I. In all 16 tests,

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TABLE I

Compression-decompression

Expt. No. (wt., kg.)	Sign	Ground	75 p.s.i.	PCG*	G+*	Time of max. effect (min.)	Altitude	Simulated height (K ft.)	G+ PCG/G+		Alt (PCG/Alt)	
									RR	MPAP	RR	MPAP
1 (8.3)	PAP	12/5	6/1	4.7	10.3	+29	55.3	18	1.44	2.19	2.67	11.78
	SAP	158/118	190/140	178/133	160/115		170/125					
	RR	16		18	28		48					
	HR	160		168	162		162					
2 (18.0)	PAP	29/8	22/10	18.7	22.0	+87	52.3	18	1.09	1.18	2.0	2.8
	SAP	168/132	190/132	176/150	156/133		159/146					
	RR	16	18	22	24		44					
	HR	192	174	210	210		154					
3 (13.2)	PAP	29/11	17/13	19.7	32.7	+33	65.7	18	.93	1.66	3.2	3.33
	SAP	150/110	160/119	162/122	150/110		165/120					
	RR	13	12	15	14		48					
	HR	140	128	150	128		156					
4 (9.1)	PAP	21/12	23/11	19.7	29.7	+29	48.0	13	1.43	1.51	1.67	2.44
	SAP	150/115	168/131	158/123	150/115		105/65					
	RR	34	36	42	60		70					
	HR	204	200	210	220		198					
5 (10.9)	PAP	22/8	20/6	13/3	28.0	+30	39.3	15.5	1.41	1.96	7.06	2.96
	SAP	168/104	150/120	190/140	140/100		140/100					
	RR	7	10	17	24		120					
	HR	160	190	210	160		140					
6 (10.0)	PAP	21/11	26/14	20.7	45.3	+31	58.3	12	3.5	2.19	3.75	2.82
	SAP	110/80	135/105	150/124	152/130		80/55					
	RR	11	11	16	56		60					
	HR	100	114	120	96		120					

4

TABLE I (Continued)

Compression-decompression

Expt. No. (wt., kg.)	Sign	Ground	75 p.s.i.	PCG*	G+*	Time of max. effect (min.)	Altitude	Simulated height (K ft.)	G+ PCG/G+		Alt (PCG/Alt)	
									RR	MPAP	RR	MPAP
7 (23.6)	PAP SAP RR HR	25/17 145/90 16 150	24/19 146/115 18 144	27.3 162/125 26 156	40.0 157/105 22 162	+27	58.7 160/100 72 156	12.5	.85	1.48	2.77	2.15
8 (20.4)	PAP SAP RR HR	30/14 160/115 6 150	19/12 150/115 5 150	15.7 165/135 15 150	69.0 205/150 38 180	+7	61.0 110/70 16 180	12	2.54	4.39	1.07	3.89
9 (12.7)	PAP SAP RR HR	22/12 149/107 24 167	23/15 155/120 16 158	12.0 140/110 34 167	13.3 140/110 86 160	+10	72.3 150/110 70 100	12	1.06	1.11	2.06	6.03
10 (23.3)	PAP SAP RR HR	23/11 135/105 10 158	16/12 133/108 8 139	7.0 150/120 8 150	43.3 158/120 54 142	+28	53.3 150/120 94 154	11.5	6.75	6.19	11.75	7.62
11 (18)	PAP SAP RR HR	24/18 115/81 12 154	22/13 126/105 14 154	18.0 181/108 21 162	20.3 180/103 30 168	+21	61.3 115/80 60 143	14	.95	1.56	2.86	4.72
12 (14.8)	PAP SAP RR HR	23/17 135/107 4 144	20/12 132/110 8 116	16.7 150/125 2 128	28.0 160/125 4 114	+42	56.3 178/132 23 126	15	2.0	1.69	11.5	3.38

TABLE I (Continued)
Compression-decompression

Expt. No. (wt, kg.)	Sign	Ground	75 p.s.i.	PCG*	G+*	Time of max. effect (min.)	Altitude	Simulated height (K ft.)	G+ PCG/G+ RR	MPAP	Alt (PCG/Alt) RR	MPAP
18 (9.5)	PAP	28/18	25/15	23.3	55.3	+18	—	—	1.0	2.37	—	—
	SAP	115/97	150/115	150/130	133/97		—					
	RR	81	80	42	42		—					
	HR	190	152	186	74		—					
14 (28.6)	PAP	24/12	14/4	20.3	29.7	+18	46.7	10	2.38	1.46	6.5	2.3
	SAP	160/120	127/104	153/111	160/125		160/120					
	RR	16	8	16	38		104					
	HR	143	186	144	142		152					
15 (17.8)	PAP	28/7	18/9	11.0	48.3	+23	62.3	10	8.05	4.39	6.87	5.67
	SAP	198/149	179/140	179/138	144/110		127/75					
	RR	20	8	16	129		110					
	HR	164	150	150	129		112					
16 (17.8)	PAP	28/7	27/5	12.0	29.0	+15	43.3	10	3.1	2.42	3.3	3.61
	SAP	173/126	178/142	183/150	122/92		140/93					
	RR	16	18	20	62		66					
	HR	150	158	176	130		—					
Average mean pulmonary artery pressure (mm. Hg)				15.9	35.1		55.6					

PAP — Pulmonary artery pressure.
SAP — Systemic artery pressure.
RR — Respiratory rate.
HR — Heart rate.
MPAP — Mean pulmonary artery pressure.
PCG — Postcompression ground control.
G+ — Ground plus.
Alt — Altitude.
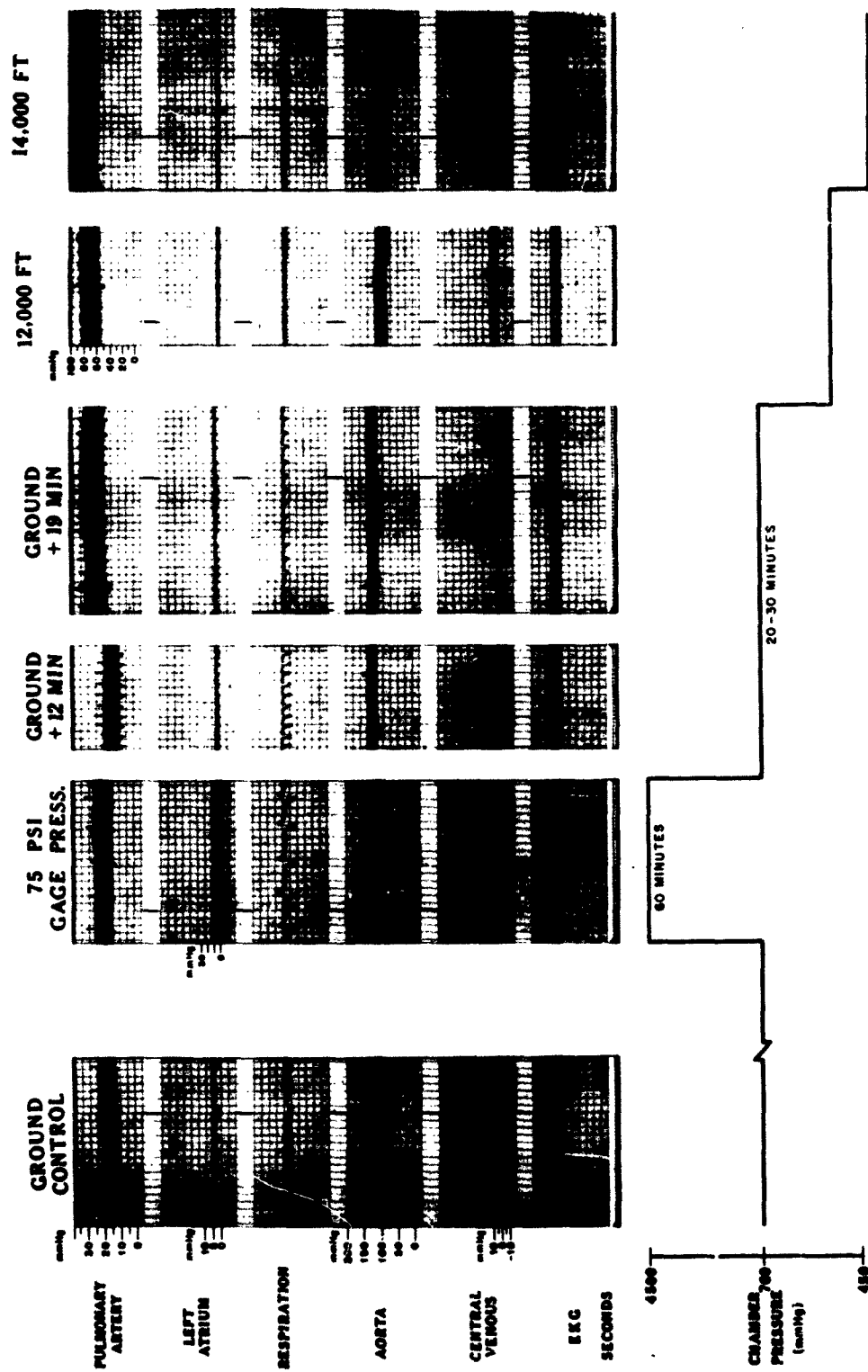*Pulmonary artery pressure expressed as mean pressure.

FIGURE 2

Typical recording of events during a compression experiment. The attenuation setting on the amplifier for pulmonary artery pressure was changed during the altitude portion of the run when recording went off scale.

changes in any dynamic measurement are listed in the two final columns labeled "ground +" and "altitude" and are compared with the post-compression ground-control period immediately following the 60-minute exposure to 6 atmospheres absolute pressure (75 p.s.i. gage pressure). A typical recording is shown in figure 2. After returning to 1 atmosphere absolute pressure, all animals developed a shallow, rapid respiration and a rapid rise in pulmonary artery pressure, both systolic and diastolic. Within 7 to 42 minutes (average 26 minutes), the average mean pulmonary artery pressure in the 16 animals rose from 15.9 mm. Hg to a maximum of 35.1 mm. Hg, or 2.2 times. During the same time, respiratory rate increased 2.4 times on the average. The unexpected decreased respiratory rate in experiments 3, 7, and 11 was probably due to maintenance doses of anesthesia given shortly after arriving at ground level. All other measurements during this period remained in the normal range as compared with the controls. It is interesting to note that, in spite of the large increase in pulmonary artery pressure, the central venous pressure remained unchanged as did the left atrial pressure in the four successful catheterizations. When the pulmonary artery pressure had stabilized at its new peak, the animal was placed on 100% oxygen and exposed to subatmospheric conditions. This was done to increase the size of bubbles—if, in fact, they were present—in the intravascular spaces. The pulmonary artery pressure rose further during the ascent to altitude, averaging 55.6 mm. Hg mean compared with the 15.9 mm. Hg postcompression value. Also, the respiratory rate was 3.5 times greater. Again, all other measurements remained in the normal range for a short period. Soon after the tremendous increase in pulmonary arterial pressure, however, the systemic arterial pressure rapidly began to fail, presumably due to a failing cardiac output by the left heart. When this occurred, the entire preparation began failing and in most instances halted in circulatory collapse and respiratory arrest. In several preparations, ventricular fibrillation was seen to intervene during the period of circulatory collapse.

After returning to 1 atmosphere, and before the usual tachypnea and elevated pulmonary arterial pressure became evident, bubbles were seen in the venous viewing cuvette in all experiments. These were seen to migrate from the peripheral vascular bed through the polyethylene catheter and into the cuvette. In all cases at this site, the flow of bubbles was profuse; whereas, none were seen in the arterial shunts, even after pulmonary arterial pressure had begun to rise. However, when these animals were taken to simulated altitude (0.69 atmosphere in these 3 cases), bubbles began appearing in the arterial shunt cuvettes in large quantities. It was at this point that the animals began showing evidence of circulatory failure with death intervening. Before the bubbles appeared in the arterial cuvettes, all dynamic measurements were in the normal range except for the large increase in pulmonary artery pressure, tachypnea, and bubbles in the venous cuvette.

Decompression series

In this series of 5 tests, 3 to .20 minutes after reaching 0.18 atmosphere pressure (40,000 ft. equivalent), small bubbles began flowing across the venous cuvette and tended to coalesce into large bubbles (fig. 3). It was easy to observe bubbles migrating across the viewing cuvette and thence into the central venous circulation. *In all cases, bubbles began flowing across the cuvette prior to any change in pulmonary arterial pressure or in respiratory rate.* Within a short time, however, pulmonary artery pressure began rising with the diastolic phase reaching an average maximum of 142.7% greater than the ground-level control. Also, respiratory rate increased an average of 368.8%. In experiment 20, in which bubbles appeared in both the arterial and venous cuvettes, pulmonary artery diastolic pressure was 137.5% greater than control; whereas, in experiment 21, in which no bubbles were noted in the arterial cuvette, pulmonary artery diastolic pressure increased only 75% (table II). When the animals were returned to ground level, the large bubbles disappeared from the cuvettes, leaving only a layer of fine bubbles at the upper edges. At the completion

FIGURE 3

Vein-to-vein (left) and artery-to-artery (right) cuvettes at 40,000 ft. equivalent altitude in an intact animal. This view was photographed through one of the observation windows in the chamber. Note bubbles just beginning to appear in the arterial cuvette.

TABLE II
Decompression - 40,000 ft. equivalent

Experiment No.	Sign	Ground control	40,000 ft. equivalent	Percent increase	
				PAP (diastolic)	RR
17	PAP	25/15	30/27	80.0	
	SAP	155/117	163/120		
	RR	8	60		650.0
	HR	124	116		
18	PAP	22/11	21/20	81.9	
	SAP	165/135	112/82		
	RR	25	78		212.0
	HR	231	176		
19	PAP	20/14	70/60	339.0	
	SAP	147/120	155/113		
	RR	34	92		170.8
	HR	156	164		
20	PAP	26/8	22/19	137.5	
	SAP	162/120	175/103		
	RR	9	40		344.5
	HR	159	146		
21	PAP	29/8	17/14	75.0	
	SAP	140/97	180/115		
	RR	6	34		467.0
	HR	124	146	Av. 142.7	Av. 368.8

See footnotes, table I.

of the experiment, however, foamy blood could be expressed from severed subcutaneous blood vessels and was observed in intact veins.

Direct infusion series

The results were not entirely satisfactory because of the inability to obtain pulmonary artery pressure recordings in 2 of the animals. In the single successful catheterization, the pulmonary artery diastolic pressure rose from 11 mm. Hg to 27 mm. Hg while the respiratory rate increased from 8 to 28 per minute following the infusion of 21 cc. of bubbles in the peripheral vein. In the other 2 animals, respiration rate increased from 6 and 8 per minute to 84 and 21 per minute, respectively. At the same time, right intraventricular systolic pressure rose from 27 mm. Hg in the former and 28 mm. Hg in the latter to 60 mm. Hg and 40 mm. Hg after the infusion of 30 and 47 cc. of bubbles in each case. The latter animal was exposed to a pressure equivalent of 0.69 atmosphere (10,000 ft.) after the bubbles were infused and after the rise in right ventricular systolic pressure was demonstrated. At this altitude, the pulmonary arterial systolic pressure (as expressed by right intraventricular systolic pressure) increased to 52 mm. Hg while the respiratory rate rose to 70 per minute.

4. DISCUSSION

The initial goal anticipated as a result of the research outlined in this paper was to establish a definite set of signs in an experimental animal which is entirely reproducible. These signs would be definite indicators that the animal, in fact, was suffering from decompression sickness and these signs would not be masked by other complications such as hypoxia or cortical influences. To this end, the compression-decompression schedule was established. Earlier, Behnke (7) reported that dogs compressed to 5 atmospheres absolute pressure for 4 hours showed no change in respiratory rate. However, if they were compressed to 6 atmospheres absolute pressure for 1½ hours and then decompressed, a typical tachypnea resulted. He described this tachypnea as pathognomonic of bubbles in the

pulmonary vessels. Megibow et al. (8, 9) measured respiration and pulmonary artery pressure in unanesthetized dogs and found a rise in pulmonary artery pressure and a tachypnea when starch granules were injected into the right heart. Binger et al. (10) injected suspensions of potato starch (5 to 60 μ in size) and obtained a typical tachypnea. Niden and Aviado (11) injected glass beads (60 to 420 μ) into the right ventricle of dogs and produced a tachypnea which they thought was mediated through the vagus nerves. They also produced evidence that these beads passed through arteriovenous shunts in the pulmonary vascular bed when the pulmonary arterial pressure rose.

In our experiments, intravascular bubbles were produced in every case in the compression-decompression series. By using the vein-to-vein and artery-to-artery cuvettes, the site of origin of the small bubbles could be located in the vascular system. The cardinal signs of decompression sickness appear to be a tachypnea and a rise in pulmonary arterial pressure. From experiments 14, 15, and 16, in which artery-to-artery cuvettes were also used, the bubbles did not appear on the arterial side until the subatmospheric exposure was accomplished following return from high positive pressure. In other words, even though pulmonary artery pressure rose during the decompression period, this rise may not have been sufficient to open the so-called labile arteriovenous shunts in the pulmonary bed. Upon reaching altitude, it was shown in all cases that pulmonary artery pressure rose even higher. This rise is probably due to further expansion of the bubbles trapped in the pulmonary bed, leading to an increased resistance to flow and a further rise in pressure.

It is interesting to note that the pulmonary hypertension did not lead to a subsequent rise in central venous pressure. A possible explanation could be that the right heart increased its force of contraction sufficiently to maintain a normal flow through the more resistant bed. This is borne out by the fact that systemic arterial pressure was maintained at a normal level as was left atrial pressure.

The delay in the signs typical of decompression sickness (tachypnea and rising pulmonary artery pressure) upon returning to 1 atmosphere was shown to correlate with the delay in bubble formation. None of the signs appeared until after many bubbles began flowing through the venous cuvette. The same delay was noted by Behnke (7) using respiratory rate as his criteria for pulmonary vascular obstruction.

As a result of these experiments, we believe that all persons exposed to altitudes greater than 23,000 ft. for even a relatively short period of time probably form intravascular bubbles, especially in the absence of denitrogenation procedures. Such bubbles could form in the periphery and migrate to the right heart where, as a result of systolic contraction, they could break again into smaller bubbles or be redissolved in the blood. Some of these bubbles then could arrive in the pulmonary circulation and be released through the normal diffusion mechanism into the lung air with no symptoms resulting. If the rate of formation of bubbles in the periphery were to increase greatly, however, it is possible that the formation rate would exceed the diffusion rate of the pulmonary circulation and that bubbles would mechanically begin to block capillary and arteriolar flow. When such a block occurs, the pulmonary vascular resistance may increase enough to cause both a rapid rise in pulmonary arterial pressure and a pronounced tachypnea, as others have shown by injecting artificial emboli in the pulmonary bed. The rises in pulmonary artery pressure and respiratory rate are aggravated to an even greater extent when animals returned from 6 atmospheres absolute pressure are then taken to subatmospheric pressures; such rises seem to verify the location of bubbles in the pulmonary bed. The further rise in both events is interpreted as a growth or expansion of the bubbles at subatmospheric pressures, causing further restriction of flow channels in the pulmonary bed and causing a greater mechanical stimulation

to unknown receptors whose nervous pathways may perhaps lie in vagal afferent fibers.

With the onset of circulatory failure, the entire pattern suddenly changes to one of impending collapse. Systemic arterial pressure begins to decline; respiration becomes slower, deeper, and more labored; pulmonary artery pressure decays; subsequently, the animal rapidly expires. The question of whether circulatory failure is due to a blockage of the pulmonary vascular pathways, or due to an "air lock" forming in the right ventricle, has arisen in the past. Likewise, the effect of mechanical stimulation by emboli in the pulmonary circulation leading to reflex vascular spasm has also been mentioned by others. Although this could be the total cause of circulatory failure, the initial symptoms are believed to be due primarily to intravascular bubbles blocking more and more of the pulmonary circulation. The eventual failure then could be one or a combination of all the three events, since, in every case, profuse bubbles were noted in the right ventricle. In our opinion, the primary problems of verifying the cause of the tachypnea and the rise in pulmonary artery pressure have been accomplished. If bubbles do cause blockage of the pulmonary bed, then overcompression should result in immediate relief. That some cases of collapse are delayed until after the subject has reached ground level is well known. It should be noted that all pictures of bubbles photographed *in situ* by Armstrong (6) were taken at ground level following altitude exposure. In our own experience, the 5 animals with bubbles evoked by exposure to 0.18 atmosphere pressure still retained some intravascular bubbles after being returned to ground level. Evidently, once a bubble is formed, it is difficult to drive it back into solution unless greater pressures are used. A recent clinical case in which high positive pressure was used in the treatment of decompression sickness tends to verify this approach (15).

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Unclassified Report

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1. Aerospace medicine
2. Altitude sickness

I. AFSC Task 775802
II. S. D. Leverett, Jr.,
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Unclassified Report

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